

Dear editors and experts:

First of all, on behalf of all the authors of the paper "Cauda equina nerve calcification: A case report" (NO.80543), I would like to express my sincere gratitude to you for your valuable suggestions on the revision of this paper. I have carefully revised the format and content of this manuscript according to the comments of editors and peer review experts. Since I am not a native speaker of English, I entrusted a professional organization to polish the language of the whole manuscript. In this modification note, I will mark the experts' comments and questions in blue font, and mark my replies and modifications in red font. Specific amendments and responses to expert opinions are as follows:

1、Add "Post-traumatic Cauda equina calcification" to the title and keywords, because the basis for the development of the disease is described in the anamnesis.

Answer: I think the expert's opinion is very constructive. I have changed the title of the article to "Post-traumatic Cauda equina calcification". The patient had a history of violent trauma 35 years ago and underwent emergency surgery. In the recent imaging data of the patient, it can also be clearly observed that the patient's spine has undergone huge pathological changes due to violent trauma. In consideration of the patient's trauma history, existing symptoms, imaging data and possible mechanism of nerve calcification, "Post-traumatic Cauda equina calcification" should be added to the title and key words. Only in this way can the relationship between trauma and cauda equina nerve calcification be reflected, and the title and content of the article be closely linked. This can not only reflect the cause of cauda equina nerve calcification, but also help to clarify the mechanism of cauda equina nerve calcification. Changing the title of the article to "Post-traumatic Cauda equina nerve calcification" can make the whole article more coherent and have more clinical re

ference significance.

2、 There are repeated sentences "Nerve calcification is usually found in the cerebral and dental nerves, and the cause is usually degenerative disease, infection, etc[3]. Nerve calcification is usually found in the cerebral and dental nerves, and the cause is usually degenerative disease, infection, etc[3]. The cause is usually degeneration, infection, etc" and "It has also been suggested that IBCG is associated with carbonic anhydrase II deficiency. The IBCG has also been suggested to be associated with carbonic anhydrase II deficiency[12][13]".

Answer: Thank you for your correction. This low-level error was caused by my careless writing. I have completely corrected such errors in the full text and deleted the duplicate statements.

3、 In the pathogenesis of the development of calcification, the arachnoiditis present during the injury has not been discussed, because the search query in pubmed also reflects the issue of Cauda equina calcification from this perspective (<https://pubmed.ncbi.nlm.nih.gov/?term=Cauda%20equina%20calcification>)

Answer: Thank the experts for their additional comments and references. Since the calcification of the cauda equina nerve in this patient is secondary to traumatic stress, it is very necessary to discuss the arachnoiditis after trauma. We searched databases such as PubMed, Web of Science and CNKI with the keywords of "cauda equina nerve calcification" and "arachnoiditis". We found about 25 articles related to cauda equina nerve calcification and arachnoiditis, and thought and extracted valuable papers. Finally, we added the content to the paper. We have added the following:

Since cauda equina nerve calcification in this patient is secondary to trauma stress, it is necessary to discuss arachnoiditis during injury. The

re are also some literatures that study the problems related to cauda equina nerve calcification from this perspective. In 1999, I. G. Bilgen reported a case of cauda equina syndrome caused by ankylosing spondylitis, a patient with adhesive arachnoiditis. CT data of the patient showed signs of dural calcification. His MRI data showed that the cauda equina had adhesions with the dorsal arachnoid membrane. They believe that arachnoiditis is responsible for cauda equina syndrome and curvilinear dural calcification^[17]. In 2021, Dr. Anna Brunner et al. introduced a case of intradural calcification caused by chronic adhesive arachnoiditis and performed a dural incision exploration, and found nerve calcification structures in the dural sac. Anna Brunner et al. believed that the main cause of calcification was the inflammation of arachnoid membrane. Arachnoid inflammation leads to excessive proliferation of arachnoid cells, which leads to excessive production of collagen tissue and rapid formation of intradural scars. Subsequently, osteoblasts are generated, leading to progressive intradural ossification. However, the pathogenesis of ossifying arachnoiditis has not been completely clarified 错误,未找到引用源。. Abad Cherif El Asri et al. reported a rare case of post-traumatic Arachnoiditis ossificans of the cauda equina nerve. Abad Cherif El Asri et al. clearly proposed that trauma、surgery、infection and subarachnoid hemorrhage are the main causes of post traumatic arachnoiditis ossificans of the cauda equina nerve 错误,未找到引用源。错误,未找到引用源。.

4、For addition, it is possible to add a pathogenesis scheme describing the mechanism of Cauda equina calcification

Answer: In the original text, we did not systematically discuss the specific mechanism of cauda equina nerve calcification after trauma, which made the paper empty and without scientific value. Therefore, we carefully supplemented the relevant mechanism of cauda equina nerve calcification after trauma according to expert opinions, and speculated and a

nalyzed the cause of cauda equina nerve calcification in this patient. The mechanism of cauda equina nerve calcification after trauma is as follows:

4.1、However, pathological calcification of cauda equina nerve is extremely rare. We speculate that cauda equina nerve calcification in this patient may be secondary to acute trauma and surgical scar, thus stimulating local lesions. In addition, lumbar spinal stenosis, lumbar disc herniation, spinal tumors and spinal infection are also possible causes of cauda equina nerve calcification 错误:未找到引用源。.

4.2、The cauda equina nerve is easy to be damaged (for example, the free nucleus pulposus and epidural hematoma will cause the cauda equina nerve to be compressed), because the cauda equina nerve has no protective sheath of connective tissue and is particularly sensitive to compression. The recovery after peripheral nerve injury is very slow, which may be due to the lack of blood supply. The nutrition of cauda equina nerve also comes from cerebrospinal fluid and blood, and the recovery of cauda equina nerve after injury is slower than that of peripheral nerve. Therefore, we can boldly speculate that the mechanism of cauda equina nerve calcification may be that the surrounding tissues of cauda equina nerve suffer from mechanical compression or tissue necrosis, which affects the blood supply of cauda equina nerve. Due to long-term poor blood supply, nerve dehydration and poor calcium and phosphorus metabolism lead to calcium salt deposition, which eventually leads to cauda equina nerve calcification[7,8].

4.3、It is worth noting here that the ESR, C-reactive protein, RF and ANA of the cases reported by us are higher than the normal values. This is consistent with the study of IBCG.

4.4、The benign tumor of the cauda equina nerve may also lead to the calcification of the cauda equina nerve. We call this benign tumor the neurilemmoma of the cauda equina nerve. spinal cord neurilemmoma

often occur in the spinal cord nerve roots, but only a few occur in the cauda equina nerve. Because the neurilemmoma of the cauda equina nerve has good activity and broad intradural space, it usually has no obvious pain symptoms. The report of neurilemmoma calcification is extremely rare. In 2012, Dr. Seung Jae Hyun and others from South Korea reported that a 21 year old patient with dystrophic neurilemmoma of cauda equina had calcification, and the neurilemmoma was completely removed by surgery. However, this report does not explain the relationship between trauma and calcification of cauda equina neurilemmoma^[12]. Paraganglioma is a neuroendocrine tumor located outside the adrenal gland. Paragangliomas originating from filum terminale or cauda equina nerve are rare (accounting for 1% of all paragangliomas and 3-4% of all tumors in the lower lumbar spine). Calcification of cauda equina nerve paraganglioma is very rare. It is noteworthy that Professor M Vural once reported cauda equina paraganglioma with obvious calcification characteristics, but did not clarify its cause and pathogenesis^[13]. Professor J Rot é s Querol once reported a case of cauda equina syndrome caused by ankylosing spondylitis, which caused calcification of lumbosacral meninges. However, the CT images of the cases reported by Professor J Rot é s Querol showed perispinal calcification rather than central calcification^[14]. Intradural lumbar disc herniation is another important cause of intraspinal calcification and even cauda equina nerve calcification. Intradural lumbar disc herniation means that the free nucleus pulposus punctures the fibrous ring, posterior longitudinal ligament and dura mater, and directly compresses the spinal cord or nerve after entering the subdural cavity, resulting in acute spinal cord or cauda equina nerve injury. Intradural disc herniation is a rare type of disc herniation, with an incidence rate of 0.26% - 0.3%. The most common location of the disease was the lumbar spine, accounting for 92%^[15]. Surgical exploration is the only standard for the diagnosis of intradural lumbar disc herniation. Although i

It is difficult to make a definite diagnosis of intradural lumbar disc herniation through imaging and symptoms, some special signal signs shown by imaging data can well indicate the existence of free intradural nucleus pulposus. For example, the Y sign refers to the prominent nucleus pulposus separating the dura and arachnoid membranes into two lines in a "Y" shape. Over time, the free nucleus pulposus entering the dura will directly compress the cauda equina nerve and calcification will occur to some extent^{错误!未找到引用源。}. We speculate that the calcification of the free nucleus pulposus will probably cause the calcification of the cauda equina nerve compressed by it.

5、Make references to sources of literature in the text of the manuscript according to the requirements of the journal.

Answer: The citation of references can reflect the rigorous academic spirit of the author and is an important basis for evaluating the academic level of the paper. References are the relevant literature information resources cited for writing papers, the carrier of academic inheritance and academic ethics, and also reflect the context of academic development.

Therefore, references are an important part of academic papers, and must also appear in the paper. I have quoted the source of literature in the manuscript text according to the requirements of the journal and in accordance with the principles of authority, timeliness, relevance, accuracy and objectivity of literature citation. The details are as follows:

[1] Joshi A, Chitale N, Phansopkar P. The Impact of Physical Therapy Rehabilitation on Pain and Function in a Patient With Cauda Equina Syndrome. *Cureus*. 2022 Aug 18;14(8):e28131. [PMID: 36134093 DOI: 10.7759/cureus.28131]

[2] Fukatsu S, Ogihara S, Imada H, Ikemune S, Tamaru JI, Saita K. Chronic spontaneous epidural hematoma in the lumbar spine with cauda equina syndrome and severe vertebral scalloping mimicking a spinal t

umor: a case report. *BMC Musculoskelet Disord*. 2022 May 30;23(1):508. [PMID: 35637479 DOI: 10.1186/s12891-022-05463-x.]

[3] Isen DR, Gaddamanugu S, Kline LB. Optic Nerve Sheath Calcification. *Ophthalmology*. 2022 Jun 27:S0161-6420(22)00208-1.[PMID: 35773073 doi: 10.1016/j.opthta.2022.03.013.]

[4] Nasim O, Eskander B, Rustam Z, Pantelias C, Moverley R. Expediting the Management of Suspected Cauda Equina Syndrome (CES) in the Emergency Department Through Clinical Pathway Design at a District General Hospital: A Quality Improvement Project. *Cureus*. 2022 Dec 20;14(12):e32722.[PMID: 36570114 DOI: 10.7759/cureus.32722.]

[5] Ando T, Watanabe H, Riku Y, Yoshida M, Goto Y, Ando R, Fujino M, Ito M, Koike H, Katsuno M, Iwasaki Y. Neurogenic intermittent claudication caused by vasculitis in the cauda equina: an autopsy case report. *Eur Spine J*. 2022 Nov 23.[PMID: 36416968 DOI: 10.1007/s00586-022-07458-7.]

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[7] Rascón-Ramírez FJ. Spinal cord stimulation and cauda equina syndrome: Could it be a valid option? A report of two cases. *Neurocirugía (Astur : Engl Ed)*. 2022 Mar-Apr;33(2):90-94. [PMID: 35248303 DOI: 10.1016/j.neucie.2020.12.002]

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[9] Li M, Fu Q, Xiang L, Zheng Y, Ping W, Cao Y. SLC20A2-Associated Idiopathic basal ganglia calcification (Fahr disease): a case family re

port. BMC Neurol. 2022 Nov 17;22(1):438.[PMID: 36397039 DOI: 10.1186/s12883-022-02973-y]

[10] Durante A, Audino N, Cristiano M, Tanga M, Martino MT, No schese I, D'Auria D, Pinto F. Basal ganglia calcification: a Fahr's disease case report. Radiol Case Rep. 2021 Aug 12;16(10):3055-3059.[PMID: 34429801 DOI: 10.1016/j.radcr.2021.07.042]

[11] Peters MEM, de Brouwer EJM, Bartstra JW, Mali WPTM, Koek HL, Rozemuller AJM, Baas AF, de Jong PA. Mechanisms of calcification in Fahr disease and exposure of potential therapeutic targets. Neurol Clin Pract. 2020 Oct;10(5):449-457.[PMID: 33299674 DOI: 10.1212/CPJ.0000000000000782.]

[12] Hyun SJ, Rhim SC. Giant cauda equina schwannoma with dystrophic calcifications : case report and review of the literature. J Korean Neurosurg Soc. 2012 Feb;51(2):105-8.[PMID: 22500204 DOI: 10.3340/jkns.2012.51.2.105. Epub 2012 Feb 29.]

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[PMID: 32983437 DOI: 10.1016/j.amsu.2020.08.022.]

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6、The sequential order of references at discussion should be corrected...for example, reference number 4 was followed by number 10 and reference number 8 was followed by number 14....etc.

Answer: Thank you very much for your reminding. Accurate reference is an important embodiment of the paper format. I have corrected all the wrong citation order of the reference, as follows:

INTRODUCTION : Post-traumatic cauda equina nerve calcification refers to the deposition of calcium in ten pairs of lumbosacral nerve roots below the conus medullaris, resulting in various clinical symptoms^[1]. The cauda equina nerve is the bridge between the spinal cord and the peripheral nerve, connecting the pseudounipolar neurons of the dorsal root ganglion and the spinal cord neurons. Because of its special anatomical structure (only a layer of nerve inner membrane, lack of corresponding protective tissue), it is vulnerable to mechanical injury. The nutrition of cauda equina nerve comes from cerebrospinal fluid and blood, and there is a relatively anemia area in the anastomosis between the spinal a

artery and the root artery supplying the cauda equina nerve, so the cauda equina nerve is more prone to decompensation due to mechanical compression^{错误!未找到引用源。}. Post-traumatic cauda equina nerve calcification is extremely rare clinically, and its etiology, pathogenesis, treatment, and prognosis are unclear. No studies or reports on Post-traumatic cauda equina nerve calcification have been inquired, and a case of Post-traumatic cauda equina nerve calcification is reported here for reference.

DISCUSSION:

Nerve calcification usually occurs in the cranial nerve and the dental nerve. It is generally accepted that nerve calcification is divided into two main types: physiological and pathological calcification. Physiological calcification is due to the natural formation of calcified spots on the nerve with age. Pathological calcification is caused by degenerative diseases, parasitic infections, ischemic necrosis of the surrounding tissue of the nerve, oxidative stress, and apoptosis^{错误!未找到引用源。}. However, pathological calcification of cauda equina nerve is extremely rare. We speculate that cauda equina nerve calcification in this patient may be secondary to acute trauma and surgical scar, thus stimulating local lesions. In addition, lumbar spinal stenosis, lumbar disc herniation, spinal tumors and spinal infection are also possible causes of cauda equina nerve calcification^{错误!未找到引用源。}. L2-5, S1-5 and the 10 pairs of nerve roots emanating from their caudal segments together form the cauda equine nerve. The cauda equina nerve, in the usual sense, is the nerve root below L2 that innervates the pelvis as well as the perineum. It is a very important nerve in the human body^{错误!未找到引用源。}. Once the cauda equina nerve undergoes pathological changes, it will lead to dysfunction of sensory nerve, motor nerve, autonomic nerve, etc., thus causing other body dysfunction. As opposed to the cauda equina nerve calcification, Cauda equina injury is relatively common in clinical practice^{错误!未找到引用源。}. The clinical symptoms of cauda equina compression were first reported by Verbiest in 1949 and were named

Cauda Equine Syndrome (CES). The clinical symptoms of cauda equina nerve injury mainly include Low back pain, numbness of one or both lower limbs, weakness of lower limbs, weakness or disappearance of tendon reflex, sensory abnormalities in the saddle region, sphincter dysfunction, urinary incontinence, and sexual dysfunction. The cauda equina nerve is easy to be damaged (for example, the free nucleus pulposus and epidural hematoma will cause the cauda equina nerve to be compressed), because the cauda equina nerve has no protective sheath of connective tissue and is particularly sensitive to compression. The recovery after peripheral nerve injury is very slow, which may be due to the lack of blood supply. The nutrition of cauda equina nerve also comes from cerebrospinal fluid and blood, and the recovery of cauda equina nerve after injury is slower than that of peripheral nerve. Therefore, we can boldly speculate that the mechanism of cauda equina nerve calcification may be that the surrounding tissues of cauda equina nerve suffer from mechanical compression or tissue necrosis, which affects the blood supply of cauda equina nerve. Due to long-term poor blood supply, nerve dehydration and poor calcium and phosphorus metabolism lead to calcium salt deposition, which eventually leads to cauda equina nerve calcification^{[7][8]}.

There are many reports on Idiopathic Basal Ganglia Calcification (IBGC) in the existing literature, but the specific cause is still unclear, and scholars believe that it is related to the following factors: (1) Genetic studies show that IBCG patients have autosomal recessive or dominant inheritance. (2) Vitrification during atherosclerosis can lead to calcium deposition, so some scholars believe that local calcium accumulation is related to changes in vascular osmotic pressure. (3) The abnormal level metabolism of iron and calcium phosphate, especially the decrease of serum ferritin level and iron binding capacity, will also lead to IBGC. (4) Exogenous toxic substances continuously stimulate and activate glutamate receptor, thus producing neurotoxin effect, leading to calcium ion deposition.

(5) It has been reported in the literature that during brain biopsy of patients with IBGC, there are immune inflammatory cells infiltrating around calcification points and ESR is accelerated, while C-reactive protein, rheumatoid factor (RF) and antinuclear antibody (ANA) are significantly increased. So the occurrence of IBGC is also related to the immune system. It is worth noting here that the ESR, C-reactive protein, RF and ANA of the cases reported by us are higher than the normal values. This is consistent with the study of IBGC. (6) The IBGC has also been suggested to be associated with carbonic anhydrase II deficiency. Regrettably, the epidemiological characteristics and specific treatment methods of basal ganglia calcification have not been studied^{[9],[10],[11]}.

The benign tumor of the cauda equina nerve may also lead to the calcification of the cauda equina nerve. We call this benign tumor the neurilemmoma of the cauda equina nerve. spinal cord neurilemmoma often occur in the spinal cord nerve roots, but only a few occur in the cauda equina nerve. Because the neurilemmoma of the cauda equina nerve has good activity and broad intradural space, it usually has no obvious pain symptoms. The report of neurilemmoma calcification is extremely rare. In 2012, Dr. Seung Jae Hyun and others from South Korea reported that a 21 year old patient with dystrophic neurilemmoma of cauda equina had calcification, and the neurilemmoma was completely removed by surgery. However, this report does not explain the relationship between trauma and calcification of cauda equina neurilemmoma^[12]. Paraganglioma is a neuroendocrine tumor located outside the adrenal gland. Paragangliomas originating from filum terminale or cauda equina nerve are rare (accounting for 1% of all paragangliomas and 3-4% of all tumors in the lower lumbar spine). Calcification of cauda equina nerve paraganglioma is very rare. It is noteworthy that Professor M Vural once reported cauda equina paraganglioma with obvious calcification characteristics, but did not clarify its cause and pathogenesis^[13]. Professor J Rot é s Que

rol once reported a case of cauda equina syndrome caused by ankylosing spondylitis, which caused calcification of lumbosacral meninges. However, the CT images of the cases reported by Professor J Rot é s Querol showed perispinal calcification rather than central calcification^[14]. Intradural lumbar disc herniation is another important cause of intraspinal calcification and even cauda equina nerve calcification. Intradural lumbar disc herniation means that the free nucleus pulposus punctures the fibrous ring, posterior longitudinal ligament and dura mater, and directly compresses the spinal cord or nerve after entering the subdural cavity, resulting in acute spinal cord or cauda equina nerve injury. Intradural disc herniation is a rare type of disc herniation, with a incidence rate of 0.26% - 0.3%. The most common location of the disease was the lumbar spine, accounting for 92%^[15]. Surgical exploration is the only standard for the diagnosis of intradural lumbar disc herniation. Although it is difficult to make a definite diagnosis of intradural lumbar disc herniation through imaging and symptoms, some special signal signs shown by imaging data can well indicate the existence of free intradural nucleus pulposus. For example, the Y sign refers to the prominent nucleus pulposus separating the dura and arachnoid membranes into two lines in a "Y" shape. Over time, the free nucleus pulposus entering the dura will directly compress the cauda equina nerve and calcification will occur to some extent 错误!未找到引用源。. We speculate that the calcification of the free nucleus pulposus will probably cause the calcification of the cauda equina nerve compressed by it.

Since cauda equina nerve calcification in this patient is secondary to trauma stress, it is necessary to discuss arachnoiditis during injury. There are also some literatures that study the problems related to cauda equina nerve calcification from this perspective. In 1999, I. G. Bilgen reported a case of cauda equina syndrome caused by ankylosing spondylitis, a patient with adhesive arachnoiditis. CT data of the patient showed sig

ns of dural calcification. His MRI data showed that the cauda equina had adhesions with the dorsal arachnoid membrane. They believe that arachnoiditis is responsible for cauda equina syndrome and curvilinear dural calcification^[17]. In 2021, Dr. Anna Brunner et al. introduced a case of intradural calcification caused by chronic adhesive arachnoiditis and performed a dural incision exploration, and found nerve calcification structures in the dural sac. Anna Brunner et al. believed that the main cause of calcification was the inflammation of arachnoid membrane. Arachnoid inflammation leads to excessive proliferation of arachnoid cells, which leads to excessive production of collagen tissue and rapid formation of intradural scars. Subsequently, osteoblasts are generated, leading to progressive intradural ossification. However, the pathogenesis of ossifying arachnoiditis has not been completely clarified 错误;未找到引用源。. Abad Cherif El Asri et al. reported a rare case of post-traumatic Arachnoiditis ossifications of the cauda equina nerve. Abad Cherif El Asri et al. clearly proposed that trauma, surgery, infection and subarachnoid hemorrhage are the main causes of post traumatic arachnoiditis ossifications of the cauda equina nerve 错误;未找到引用源。错误;未找到引用源。.

Since cases of Post-traumatic cauda equina nerve calcification are extremely rare in clinical practice, our research on this disease has not been carried out and the literature that we have been able to study is very scarce. Therefore, it is necessary that we carry out studies on the etiology, mechanisms, treatment and prognosis of this disease.

Reference.

- [1] **Joshi A**, Chitale N, Phansopkar P. The Impact of Physical Therapy Rehabilitation on Pain and Function in a Patient With Cauda Equina Syndrome. *Cureus*. 2022 Aug 18;14(8):e28131. [PMID: 36134093 DOI: 10.7759/cureus.28131]
- [2] **Fukatsu S**, Ogihara S, Imada H, Ikemune S, Tamaru JI, Saita K. Chronic spontaneous epidural hematoma in the lumbar spine with ca

- uda equina syndrome and severe vertebral scalloping mimicking a spinal tumor: a case report. *BMC Musculoskelet Disord*. 2022 May 30;23(1):508. [PMID: 35637479 DOI: 10.1186/s12891-022-05463-x.]
- [3] **Isen DR**, Gaddamanugu S, Kline LB. Optic Nerve Sheath Calcification. *Ophthalmology*. 2022 Jun 27:S0161-6420(22)00208-1.[PMID: 35773073 doi: 10.1016/j.opthta.2022.03.013.]
- [4] **Nasim O**, Eskander B, Rustam Z, Pantelias C, Moverley R. Expediting the Management of Suspected Cauda Equina Syndrome (CES) in the Emergency Department Through Clinical Pathway Design at a District General Hospital: A Quality Improvement Project. *Cureus*. 2022 Dec 20;14(12):e32722.[PMID: 36570114 DOI: 10.7759/cureus.32722.]
- [5] **Ando T**, Watanabe H, Riku Y, Yoshida M, Goto Y, Ando R, Fujino M, Ito M, Koike H, Katsuno M, Iwasaki Y. Neurogenic intermittent claudication caused by vasculitis in the cauda equina: an autopsy case report. *Eur Spine J*. 2022 Nov 23.[PMID: 36416968 DOI: 10.1007/s00586-022-07458-7.]
- [6] **Khashan M**, Ofir D, Grundshtein A, Kuzmenko B, Salame K, Nirry D, Hochberg U, Lidar Z, Regev GJ. Minimally invasive discectomy versus open laminectomy and discectomy for the treatment of cauda equina syndrome: A preliminary study and case series. *Front Surg*. 2022 Oct 12;9:1031919.[PMID: 36311945 DOI: 10.3389/fsurg.2022.1031919.]
- [7] **Rascón-Ramírez FJ**. Spinal cord stimulation and cauda equina syndrome: Could it be a valid option? A report of two cases. *Neurocirugía (Astur : Engl Ed)*. 2022 Mar-Apr;33(2):90-94. [PMID: 35248303 DOI: 10.1016/j.neucie.2020.12.002]
- [8] **Moussa MK**, Alkefrawi P, Elkhailil JK. Large central disc herniation causing cauda equina syndrome in an adolescent. A case report. *Int J Surg Case Rep*. 2021 Feb;79:119-122.[PMID: 33454631 DOI: 10.101

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- [9] **Li M**, Fu Q, Xiang L, Zheng Y, Ping W, Cao Y. SLC20A2-Associated Idiopathic basal ganglia calcification (Fahr disease): a case family report. BMC Neurol. 2022 Nov 17;22(1):438.[PMID: 36397039 DOI: 10.1186/s12883-022-02973-y]
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In addition to the parts required by experts, I have carefully revised the whole paper in terms of format, language, content and scientificity, so as to strive to meet the publication standards of your journal. Please refer to the revised manuscript for specific contents. Here, on behalf of all the authors of the paper "Cauda equina nerve calcification: A case report" (NO.80543), I would like to thank you again for your hard work!

I wish you good health and all the best.

Yours Sincerely

Qiang Deng